"Efficiency of work production by spastic muscles"

Stoquart, Gaëtan ; Detrembleur, Christine ; Nielens, Henri ; Lejeune, Thierry

ABSTRACT

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Efficiency of work production by spastic muscles

G.G. Stoquarta, C. Detrembleura,*, H. Nielensb, T.M. Lejeuneb

a Rehabilitation and Physical Medicine Unit, Université catholique de Louvain, Tour Pasteur 5375, Avenue Mounier 53, B-1200 Brussels, Belgium
b Department of Physical Medicine and Rehabilitation, Cliniques universitaires Saint-Luc, Université catholique de Louvain, Brussels, Belgium

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Abstract

The present study compared the muscular efficiency in spastic and healthy lower limbs producing the same mechanical work. Sixteen chronic post-stroke hemiparetic and spastic patients and 14 age-matched healthy subjects were submitted to a submaximal stepwise exercise testing on a bicycle ergometer, pedalling with only one lower limb. Net energetic expenditure was computed from oxygen consumption above resting values. Electrical activity of antagonistic muscles in the thigh and in the shank was recorded and co-contraction was defined as the percentage of the pedalling cycle when antagonistic muscles were activated simultaneously. The efficiency was calculated as the ratio between the mechanical work done on the ergometer and the net energetic expenditure. Spasticity was quantitatively evaluated by measuring passive ankle plantar flexor muscle stiffness. The working capacity of the patients’ paretic lower limb was very low (<40 W). The energy expenditure increased linearly as a function of work intensity, without statistical difference between the patients paretic lower limb (PPL), the patients healthy lower limb (PHL) and the healthy subjects lower limb (HSL). Shank co-contraction was 2.9 times greater in PPL (p<0.05) and 2.3 times greater in PHL (p<0.05) than in HSL. Thigh co-contraction was also 1.8 times greater in PPL than in HSL (p<0.05). The ankle plantar flexor muscle stiffness was statistically greater in PPL than in PHL and HSL (p<0.05). The efficiency was not statistically different between the three groups (p=0.155). In conclusion, the efficiency of work production by paretic and spastic lower limb muscles was normal (≥20%) despite significant neurological impairments.

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Keywords: Stroke; Efficiency; Energetics; Mechanics; Cycling

1. Introduction

Locomotion of hemiparetic and spastic subjects requires more energy than that of healthy subjects [1]. Bernardi et al. [2] and Zamparo et al. [3] found that this increase was inversely related to the walking speed spontaneously adopted by patients, and thus to the severity of neurological impairments. Energy expenditure is determined by the mechanical work done by muscles and by the efficiency of mechanical work production by muscles. The increase in energy expenditure of walking in hemiparetic subjects must then be related to either an increased mechanical work or a decreased efficiency. The mechanical work may be increased as a result of segmental kinematic impairments such as equinus, stiff knee or increased step frequency. In spastic and paretic patients, motor control is disturbed and the efficiency may be decreased as a result of inappropriate muscle activation, i.e. muscles could expend useless energy during excessive co-contraction. Various alterations of muscle properties such as increased muscle tone or contracture, modified muscle fibre size and type, altered mechanical and morphological properties of intra- and extra-cellular material [4] may also lead to decreased efficiency. A previous study [5] conducted in nine chronic hemiparetic patients confirmed that the increase of the energy expended per metre (C, the net energetic cost, J kg⁻¹ m⁻¹) was inversely related to walking speed. C was shown to be up to two times greater than normal values in slower patients (1.7 km h⁻¹), whereas it was only 1.2 times greater in faster patients (3.6 km h⁻¹). The mechanical work, measured following the method described by Cavagna and Kaneko [6], increased proportionally to C. The efficiency then remained similar to

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* Corresponding author. Tel.: +32 2 764 53 75; fax: +32 2 764 53 60.
E-mail address: detrembleur@read.ucl.ac.be (C. Detrembleur).

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values measured in healthy subjects walking at the same speed.

During a bilateral activity, such as walking, both healthy and pathological limbs contribute to the mechanical work, energetic cost and efficiency. Hemiparetic gait is typically asymmetric with the healthy lower limb producing more work than the pathological one [7]. Therefore, the normal efficiency found by Detrembleur et al. [5] could be explained by the fact that only minimal mechanical work is done by the impaired lower limb. If the healthy lower limb performs most of the mechanical work, where muscles are working under normal conditions, then whole body efficiency should remain normal even if spasticity, contracture and co-contraction may partially decrease efficiency (η) in the impaired lower limb. The aim of the present study was to measure the energy expended by hemiparetic patients while pedalling, one lower limb at a time, on a cycle ergometer. The efficiency of work production was then compared between the hemiparetic spastic lower limb and the healthy lower limb.

2. Materials and methods

2.1. Study population

Sixteen chronic hemiparetic and spastic patients (11 men and 5 women; mean age: 58.6 ± 13.6 years; mean height: 172 ± 11 cm; mean weight: 76 ± 12 kg) were recruited from our outpatient rehabilitation unit between January and April 2003. In the hemiparetic patients the time elapsed from their stroke was 44.7 ± 54.5 months. Anthropometric and historical data are presented in Table 1. All patients were able to walk independently during daily life activities, corresponding to a score ≥4 on the Functional Ambulation Categories Scale [8]. All were also able to pedal on a cycle ergometer for more than 5 min, and then to complete a stepwise metabolic analysis and had no other major medical disorder. In the acute phase, all patients had been treated in a stroke unit and, thereafter, had followed an intensive rehabilitation program. Ten patients were still having physiotherapy twice a week to maintain their functional capacities.

Fourteen healthy age-matched subjects (eight men and five women; mean age: 59 ± 12.4 years; mean height: 173 ± 9 cm; mean weight: 76 ± 12 kg) were also evaluated and served as controls. Heights and weights were similar in the healthy subjects and patients.

All subjects participated freely in the study after giving their informed consent. The ethical board of the Université catholique de Louvain medical school approved the study.

2.2. Neurological impairments assessment

The same physician (SG) performed all clinical examinations and tests for hemiparetic and healthy subjects. Lower limb neurological impairments were evaluated by a subscale of the Stroke Impairment Assessment Set (SIAS; [9]) assessing lower limbs (maximal score: 43). Spasticity of the Triceps Surae and Quadriceps Femoris was evaluated clinically on the affected side by the modified Ashworth Scale [10]. Spasticity was also quantitatively evaluated by measuring ankle plantar flexor muscle stiffness [11,12]. This method quantified the increase of resistance to passive movement by a mechanical device. The subject was placed in a supine position with one foot attached to a mobile plate. A motor generated low amplitude sinusoidal displacements of the ankle joint (±2.5°) at different frequencies (3–12 Hz).

Table 1

<table>
<thead>
<tr>
<th>No.</th>
<th>Sex (M/F)</th>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>Paretic side (right/left)</th>
<th>Time from stroke (months)</th>
<th>SIAS score (/43)</th>
<th>Ashworth Scale score (/5)</th>
<th>FAC score (/5)</th>
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<td>172.1</td>
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<td>[24;41]</td>
<td>[2;4]</td>
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</table>

* Median and range are used instead of mean and standard deviation for non-continuous variables.
The relaxed, passively displaced calf–ankle–foot system can be modelled, in mechanical terms, as a torsional spring, torsional viscous damper and rotative mass connected in parallel. The application of sinusoidal displacement to such a passive visco-elastic system produces a characteristic torque response. The torsional spring, first element of the model, contributes a torque response (elastic stiffness, first panel of Fig. 1) in phase with the displacement, with an amplitude that is dependent on amplitude of the displacement and stiffness of the spring. The amplitude of this torque response is independent of the frequency of the sinusoidal displacement. In the present study, elastic stiffness was characterized by the \( y \)-intercept of the linear relation between elastic stiffness and movement frequencies. The second element of the model, a torsional viscous damper, produces a resisting force in proportion to the velocity applied to it and the viscosity of the damper. The torque response of the damper (viscous stiffness, second panel of Fig. 1) will be related linearly to the frequency of the displacement sinusoid. Viscous and elastic stiffness for varying frequencies of the displacement can be plotted together to yield a visco-elastic stiffness vector (third panel of Fig. 1), with elastic stiffness being the abscissa and viscous stiffness the ordinate. In healthy subjects, this stiffness vector will trace out a vertical line, viscous stiffness increasing as a function of the frequency, whereas elastic stiffness remains almost constant. For the spastic patient, the shape of the visco-elastic stiffness vector is very different, with both elastic and viscous stiffness responses varying non-linearly with frequency. The visco-elastic plot will often appear as a distorted “C”.

A single, quantitative descriptor of this visco-elastic frequency response is the path length (L-path), which is calculated from the total length of the path formed by the apex of the different total stiffness vectors, corresponding to the different frequencies of oscillation. The L-path for adults with spasticity is significantly longer than that for unaffected adults and increases with increasing gains of the reflex-loop element. The L-path is then a measure of the variation in visco-elastic stiffness over a range of frequencies. Both L-path and elastic stiffness y-intercept were analysed in the present study.

2.3. Bicycle ergometer testing

All subjects were asked to perform a submaximal stepwise exercise testing protocol on a modified cycle ergometer pedalling with only one lower limb at a time. A Monark 818E cycle ergometer (MONARK, Sweden) was modified by replacing the free chain–wheel by a fixed chain–wheel to allow easy passive upstroke when pedalling with only one lower limb. All subjects were seated on a chair fixed at the rear of the bicycle, to minimize effort related to posture. The exercising foot was attached to the pedal by a strap. The unexercising contralateral lower limb was placed on the other side of the bicycle, so that it did not interfere with the crank rotation.

The cycling exercise was assessed by a three-dimensional (3D) motion analysis system, including synchronous kinematic, electromyographic (EMG) and metabolic recordings. Segmental kinematics were measured with the Elite system (BTS, Italy) at 100 counts per second. Six infrared cameras measured the co-ordinates of five reflective markers placed on specific anatomical landmarks (acromion, greater trochanter of the hip, lateral condyle of the knee, lateral malleolus of the ankle and fifth metatarsal head) to compute the angular displacement of head–arm–trunk segment (HAT), hip, knee and ankle in the sagittal plane. One marker was placed on the contralateral pedal to allow delimitation of cycles to compute the cycling cadence used for further analysis.

![Fig. 1. Ankle plantar flexor muscle stiffness is represented for healthy subjects (black circles) and for the spastic leg of one representative patient (patient no. 3; white circles). In the two upper panels, each point represents mean elastic and viscous stiffness (N m rad\(^{-1}\)) as a function of oscillation frequency (Hz). Elastic stiffness y-intercept (y-int) is represented in the upper panel. The lower panel (phase diagram) shows the time course of viscous stiffness as a function of elastic stiffness.](image)
2.3.1. Muscle activity

The muscle electrical activity of Rectus Femoris (RF), Biceps Femoris (BF), Tibialis Anterior (TA) and Lateral Gastrocnemius (LG) muscles was recorded by a telemetric EMG system (Telemg, BTS, Italy) with surface electrodes (Medi-Trace, Graphic Controls Corporation, NY, USA). The signal was digitized at 1000 counts per second, full-wave rectified and filtered (bandwidth 25–300 HZ). The onset and cessation of muscle activity were both visually and mathematically determined by computing the EMG threshold voltage as described by Van Boxtel et al. [13]. The EMG activity of each muscle was normalized to 100% time of pedalling cycle. Five to nine cycles were then averaged. Co-contraction of each muscle was normalized to 100% time of segments relative to the body centre of mass (COMb).

2.3.2. Mechanical work

The total positive mechanical work done by the muscles ($W_{\text{int}}$) was divided in two parts [5,14]. The external mechanical work ($W_{\text{ext}}$) was defined as the work done against forces external to the system. In this experiment, the braking force of the ergometer represented the external force and $W_{\text{ext}}$ corresponded to the workload. The internal work ($W_{\text{int}}$) was defined as the work done to move the body segments relative to the body centre of mass (COMb). $W_{\text{int}}$ was computed from kinematic data following the method described by Willems et al. [14]. The body was divided into four rigid segments: HAT and exercising thigh, shank and foot. The internal mechanical energy of the body segments corresponded to the sum of the rotational and translational energies of these segments due to their movements relative to the COMb in the sagittal plane. The internal mechanical energy–time curves of the thigh, shank and foot were rectified and filtered (bandwidth 25–300 HZ). The onset and signal was digitized at 1000 counts per second, full-wave rectified and filtered (bandwidth 25–300 HZ). The onset and cessation of muscle activity were both visually and mathematically determined by computing the EMG threshold voltage as described by Van Boxtel et al. [13]. The EMG activity of each muscle was normalized to 100% time of pedalling cycle. Five to nine cycles were then averaged. Co-contraction of each muscle was normalized to 100% time of segments relative to the body centre of mass (COMb). Attachments to the pedal. The testing was then started and while the subject was sitting on the chair, with one foot

2.3.3. Energy expenditure

The energy expended was determined by the patient’s oxygen consumption ($\dot{V}O_2$). Breath by breath $\dot{V}O_2$, carbon dioxide production ($\dot{V}CO_2$) and heart rate were measured throughout the whole testing procedure (Quark b², Cosmed, Italy). Values were automatically converted by the system software to standard temperature, pressure and dry $\dot{V}O_2$ (Cosmed Quark b² win, Version 5.1a). The respiratory quotient (RQ) was computed as the ratio between $\dot{V}CO_2$ and $\dot{V}O_2$. Each energy measurement started by a resting period while the subject was sitting on the chair, with one foot attached to the pedal. The testing was then started and $\dot{V}O_2$ measured during the whole cycling test. RQ remained less than one during all tests. The energy expended at rest was subtracted from the energy expended when pedalling to obtain the net energy expenditure ($\dot{V}O_2$–net). The $\dot{V}O_2$–net was then converted to Watts assuming an energetic equivalent of 20.1 J per $\dot{V}O_2$ litre [15]. The efficiency of positive work production by muscles ($\eta$) was calculated as the ratio between $W_{\text{tot}}$ and the $\dot{V}O_2$–net.

2.3.4. Testing protocol

In patients, the pathological lower limb (PPL) and the healthy lower limb (PHL) were successively assessed, starting with the PPL in eight out of the 16 patients. Only one lower limb was assessed in healthy subjects (HSL), because of the equivalent results obtained in both lower limbs in a pilot study in four healthy subjects. After the resting period, all subjects were asked to pedal at a constant cadence of 30 revolution per minute or 40 revolution per minute. The predetermined workload was achieved by combining the cadence and the braking resistance. The recordings of kinematic and electromyographic data started once the cadence was stabilized. Because of the very limited PPL group working capacity, the test started with a very low external workload (7.5 W) and increased in small stages (from 3 to 10 W). Each test stage was continued until a $\dot{V}O_2$–net steady state was reached and maintained for at least 2 min. The next stage was then started and the workload increased while patients continued pedalling. Patients were asked to produce as many workload stages as possible, according to their physical capacity. The exercise stopped because of an elevated RQ (>1), pain or subject’s fatigue. Patients produced the same range of workload stages with both lower limbs. Control subjects were asked to develop the same workload range as the patients. When the same $W_{\text{ext}}$ was realized by the PPL, PHL or HSL, this workload was considered to be the same. The same $W_{\text{ext}}$ was used to compare the combined effects of lower limb groups (PPL, PHL and HSL). A two-way ANOVA was used to study the difference of co-contraction timing, ankle plantar flexor stiffness and $\eta$ between healthy and pathological lower limbs.

2.4. Statistical analysis

A paired t-test was used to compare co-contraction timing, ankle plantar flexor stiffness, $W_{\text{tot}}$, $\dot{V}O_2$–net and $\eta$ between healthy and pathological lower limbs. A one-way ANOVA was used to study the difference of co-contraction timing, ankle plantar flexor stiffness and $\eta$ between lower limb groups (PPL, PHL and HSL). A two-way ANOVA was used to study the combined effects of lower limb groups and $W_{\text{ext}}$ on co-contraction timing and $\eta$. A linear regression was utilized to explore the relation between $W_{\text{tot}}$ and $\dot{V}O_2$–net.

3. Results

All patients presented with neurological impairments. Lower limbs SIAS subscale results are presented in Table 1. The median for the lower limbs SIAS score was 33 (ranging from 24 to 41). Muscle tone of Quadriceps Femoris and Triceps Surae were increased in all hemiparetic limbs with Ashworth Scale scores ranging from 2 to 4 (Table 1). Results of ankle plantar flexor muscle stiffness measurements are

Table 1. Lower limbs SIAS subscale results

<table>
<thead>
<tr>
<th>Limb</th>
<th>SIAS Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>PPL</td>
<td>33</td>
</tr>
<tr>
<td>PHL</td>
<td>33</td>
</tr>
<tr>
<td>HSL</td>
<td>33</td>
</tr>
</tbody>
</table>

The median for the lower limbs SIAS score was 33 (ranging from 24 to 41). Muscle tone of Quadriceps Femoris and Triceps Surae were increased in all hemiparetic limbs with Ashworth Scale scores ranging from 2 to 4 (Table 1). Results of ankle plantar flexor muscle stiffness measurements are
reported in Table 2. The PPL group had 1.4 times greater L-path (195.6 ± 116.2 N m rad\(^{-1}\); \(p < 0.001\)) and mean elastic stiffness value (y-intercept: 118.9 ± 62.7 N m rad\(^{-1}\); \(p < 0.001\)) than the PHL group (L-path: 144.1 ± 41.2 N m rad\(^{-1}\); elastic stiffness y-intercept: 85.0 ± 38.3 N m rad\(^{-1}\)). The mean L-path in PPL group was also 1.5 times greater than the mean L-path in HSL group (130.1 ± 25.3 N m rad\(^{-1}\); \(p < 0.05\)). The mean L-path of the PHL group was not statistically different compared to the HSL group (\(p > 0.05\)).

Timing of muscle activation was also disturbed. TA–LG and RF–BF mean co-contraction timing indices are 2.4\(a\) times greater than in HSL. TA–LG co-contraction index was also 2.3 times greater (\(p < 0.05\)) in PHL (13.3 ± 11.7\%) than in HSL. No statistical difference was found between PPL and PHL for TA–LG (\(p = 0.939\)) and RF–BF (\(p = 0.180\)) co-contraction index. It must be noted that \(W_{\text{ext}}\) had no effect on TA–LG co-contraction indexes (\(p = 0.699\)), but that RF–BF co-contraction index increased in relation to \(W_{\text{ext}}\) (\(p = 0.012\)).

The physical capacity of patients was very low. The greatest \(W_{\text{ext}}\) developed by PPL and PHL was 37.5 W while four patients could only develop 7.5 W. The mean \(W_{\text{int}}\) values for all groups were 2.4 ± 0.8 W at 30 cycles per min and 2.9 ± 1.3 W at 40 cycles per min, without statistical difference between groups (\(p > 0.05\)). When plotting \(V\text{O}_2\)–net values as a function of \(W_{\text{int}}\) (Fig. 2), most of the \(V\text{O}_2\)–net values of the PPL and PHL groups were inside the \(V\text{O}_2\)–net prediction interval of the HSL group. No significant difference in \(V\text{O}_2\)–net values was found between PPL and PHL (\(p = 0.107\)). In the 12 patients who achieved two or more stages, linear regressions were obtained between \(V\text{O}_2\)–net and \(W_{\text{tot}}\). Slope (\(p = 0.664\)) and intercept (\(p = 0.152\)) were not different between PPL and PHL. Mean \(R^2\) of this linear relation was 0.98 (range: 0.86−1) in the eight patients who realized three or more workloads. Efficiency was then similar (\(p = 0.155\)) in PPL (19.1 ± 4.7\%), PHL (20.9 ± 5.3\%) and HSL (20.7 ± 4.2\%) groups (Fig. 2).

4. Discussion

4.1. Muscular efficiency and cycling

The present study shows that the efficiency of positive work production by lower limb muscles of hemiparetic and spastic subjects is normal and similar to the values reported in the literature for cycling with both lower limbs in healthy subjects (20–29\%; [16]), despite significant neurological impairments (SIAS, Ashworth Scale) and perturbation of muscle activation pattern. This efficiency is often thought to be decreased. However, it is rarely measured since it necessitates the computation of both the work done and the energy expended by the muscles. Cycle ergometer testing is the easiest way to make those measurements simultaneously. Spastic subjects are rarely submitted to cycle ergometer testing and there are only two previous reports on bicycle ergometer testing in spastic subjects. In the seventies, Lundberg [17] measured a lower \(\eta\), approximately 12\%, among young diplegic cerebral palsy (CP) adults than

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**Table 2**

<table>
<thead>
<tr>
<th>Groups</th>
<th>Mean ± S.D.</th>
<th>Efficiency (%)</th>
<th>L-path (N m rad(^{-1}))</th>
<th>Elastic stiffness (N m rad(^{-1}))</th>
<th>TA-LG co-contractions (% cycle)</th>
<th>RF-BF co-contractions (% cycle)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PPL</td>
<td>19.1 ± 4.7</td>
<td>195.6 ± 116.2(a)</td>
<td>118.9 ± 62.7(b)</td>
<td>16.8 ± 18.6(a)</td>
<td>16.0 ± 12.1(a)</td>
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<tr>
<td>PHL</td>
<td>20.9 ± 5.3</td>
<td>144.1 ± 41.2</td>
<td>85.0 ± 38.3</td>
<td>13.3 ± 11.7(a)</td>
<td>11.9 ± 14.1</td>
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<tr>
<td>HSL</td>
<td>20.7 ± 4.2</td>
<td>130.1 ± 25.3</td>
<td>94.1 ± 21.3</td>
<td>5.9 ± 9.1</td>
<td>8.9 ± 9.1</td>
<td></td>
</tr>
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</table>

PPL: patients pathological leg; PHL: patients healthy leg; HSL: healthy subjects leg.

\(a\) Indicates data statistically different between patients and controls subjects.

\(b\) Indicates data statistically different between PPL and PHL.
among healthy subjects, approximately 21%. However, workloads and pedalling rates were lower in diplegic patients than in controls, and RQ was not always <1. Patients pedalled seated on the saddle. In this position, diplegic CP patients may also expend additional energy to maintain trunk posture. At a similar workload (0.5 W kg⁻¹), Dresen et al. [18] found a normal increase of gait, the increase of on hemiplegic children, but a decreased C among tetraplegic children. It is important to note that these authors used an efficiency calculation excluding $W_{\text{int}}$. This could lead to a significant underestimation of the mechanical work done by the muscles. $W_{\text{int}}$ is mainly determined by the pedalling cadence but not by the workload ($W_{\text{ext}}$). In our study, $W_{\text{int}}$ represented from 6% to 25% of $W_{\text{tot}}$.

4.2. Muscular efficiency and walking

Efficiency is rarely assessed during pathological gait, because few researchers measure the total mechanical work done during gait. In nine chronic hemiparetic patients (median SIAS score: 65/76; range: 35–69), Detrembleur et al. [5] reported that the increase of C was proportional to the increase of $W_{\text{tot}}$. The efficiency of positive work production was normal, despite significant spasticity and co-contraction. On the contrary, when normal subjects use swing-through crutch gait, the increase of C was two times greater than the increase of $W_{\text{tot}}$ and $\eta$ was half than that in normal walking [16]. This reduced efficiency is explained by additional stabilization work to maintain the posture on crutches, by a lower efficiency of work production in upper limb muscles compared to lower limb muscles, and by a decrease of elastic energy storage and recovery in muscles and tendons.

4.3. Asymmetry of mechanical work

During pathological gait resulting from stroke or amputation, the work done by the muscles is frequently asymmetric [5,7]. The healthy lower limb generates more mechanical work than the pathological lower limb. Brown and Kautz [19] found the same pattern during cycling. Fifteen post-stroke hemiparetic subjects were asked to pedal with both lower limbs on a cycle ergometer. Mechanical work needed to move the pedal was computed from the measurement of forces exerted on pedals by strain gauges and angular rotation of pedals by optical encoders. The positive work done by the paretic limb during the downstroke was 1.4 times smaller than that of the healthy limb. Moreover, the paretic limb realized negative work during the upstroke, braking the healthy limb during its downstroke. Work done by the healthy limb was found to be normal. Using the same protocol, Kautz and Brown [20] found that the paretic limb realized six times less net mechanical work (positive minus negative work) than the healthy lower limb. Hence, subjects were asked to pedal one lower limb at a time in the present study to impose the same work to each limb and avoid any interference from one limb to the other.

4.4. Working capacity

The working capacity of PPL was smaller than HSL in this study. Varied positions on the bicycle were tested but none of them allowed the PPL to reach higher workloads. It represented an unavoidable limitation of the study. However, in similar patients, VO₂–net during walking is 1.2–2 times greater than in healthy subjects. Such a strong difference should be observable in our experiments, even at low workloads, if co-contraction, contracture and spasticity are responsible for the increase in energy expenditure. A similar protocol was used by Chin et al. [21] to evaluate the fitness of young adult traumatic amputees (mean age: 26.0 ± 5.7 years) pedalling one lower limb at a time with their healthy lower limb. The mean maximal workload that patients could reach was 67.6 ± 20.2 W, whereas healthy subjects could reach 102.4 ± 33.6 W. This low working capacity could also be explained by cardiovascular deconditioning and comorbidities [22], neurological impairments [24], muscular atrophy, decreased lean tissue mass and increased intramuscular-fat in paretic lower limb [23].

4.5. Spasticity, co-contraction and muscular efficiency

Winter [24] presented co-contraction as a possible cause for decreased $\eta$ during exercise. In nine spastic CP children, Unnithan et al. [25] analysed the relation between the energy consumption and co-contraction during walking at 3 km h⁻¹. Energy consumption increased in CP children in relation to thigh ($r^2 = 51.4\%$) and shank co-contraction ($r^2 = 42.8\%$). This result does not imply that the efficiency is decreased. The energy consumption increase could also result from mechanical work perturbations induced by co-contraction. Unfortunately, the mechanical work was not measured in their study.

From their study using a bicycle ergometer, Lundberg [17] explained the efficiency variation between cerebral palsied and control children by the excessive energy expenditure associated with involuntary local muscle activity. The decreased working capacity of the plegic lower limb found by Kautz and Brown [20] was strongly related to the prolonged excitation of the Vastus Medialis ($r = -0.875$), the phase-advanced excitation of the Rectus Femoris ($r = -0.896$) and the early termination of the semimembranous phase-advanced excitation ($r = -0.744$). The healthy limb’s EMG was found to be normal. In the present study, co-contraction did not influence VO₂–net.

4.6. Healthy lower limb of patients

Another interesting point is the co-contraction index and ankle muscle stiffness of PHL. As shown in Table 2, PHL TA–LG co-contraction index was 2.3 times greater than that of HSL group ($p < 0.05$). Moreover, RF–BF co-contraction and L-path tended to be greater than HSL values, but without reaching a statistically significant difference. The PHL
showed some signs of perturbed motor control and was not absolutely "normal". In 10 hemiparetic patients, Thilmann et al. [26] found increased EMG responses to stretch in the healthy triceps of hemiparetic patients, with shorter latency activity on the healthy side of hemiparetic subjects. The biceps presented a depression of the EMG response to stretch, with a decreased biceps tendon reflex \( p < 0.001 \). This pattern was thought to result from a modulation of agonist/antagonist activity in the ipses- and contralateral-limb at a spinal level. Other authors found the same increased spasticity in the healthy limb \[27,28\]. These ipsilateral findings may be a primary effect of the neurological lesion affecting the low percentage of pyramidal tract using unilateral pathways. Ipsilateral co-contraction may also be an adaptation for postural stability.

5. Conclusion

This study shows that the efficiency of work production by spastic muscles is not altered by co-contraction, contracture and spasticity. It strengthens the view that the energy expenditure is mainly determined by the mechanical work done by muscles, confirming results obtained in walking \[5,29\]. The increased \( \dot{V}O_2 \) in hemiparetic and spastic patients during exercise is then mainly explained by an increase of the mechanical work done by the muscles. Further research should be focalised on understanding the mechanics of pathological gait and future treatments directed on decreasing that mechanical work.

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